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Report

Different Dynamics of Performance and Brain Activation in the Time Course of Perceptual Learning

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SUMMARY

Perceptual learning is regarded as a manifestation of experience-dependent plasticity in the sensory systems, yet the underlying neural mechanisms remain unclear. We measured the dynamics of performance on a visual task and brain activation in the human primary visual cortex (V1) across the time course of perceptual learning. Within the first few weeks of training, brain activation in a V1 subregion corresponding to the trained visual field quadrant and task performance both increased. However, while performance levels then saturated and were maintained at a constant level, brain activation in the corresponding areas decreased to the level observed before training. These findings indicate that there are distinct temporal phases in the time course of perceptual learning, related to differential dynamics of BOLD activity in visual cortex.

INTRODUCTION

A central goal of neuroscience research is to establish links between behavior and underlying neural mechanisms. Perceptual learning (PL), defined as an increase in performance or sensitivity to a sensory feature as a result of repetitive training or exposure to that feature, is regarded as a manifestation of sensory plasticity (Fahle and Poggio, 2002; Gilbert et al., 2001; Karni and Sagi, 1991; Watanabe et al., 2001). A large number of studies have been devoted to examining PL in the hope of clarifying the link between improved performance of a perceptual task and the underlying plasticity (Ghose et al., 2002; Mukai et al., 2007; Schiltz et al., 1999; Schoups et al., 2001). So far, most neuroscientific approaches to studying PL have focused on clarifying which brain areas are involved and how the response properties of such area(s) change as a result of the development of PL. However, it remains unclear how sensory plasticity occurs in identified brain areas and how performance and activity change on a long-term basis.

The purpose of the present study was to examine how brain activation changes in the visual cortex over a long time course of PL. To study these changes, we used a texture discrimination task (TDT), a standard visual PL task that is known to involve V1 (Karni and Sagi, 1991; Schwartz et al., 2002; Walker et al., 2005). By measuring brain activation during a long time course of PL, we found that the dynamics of performance and brain activation in V1 differ throughout the time course of PL, suggesting that visual plasticity in this task is comprised of two distinct phases in which there is an initial increase in both performance and BOLD activation in visual cortex, followed by a performance saturation and maintenance phase, in which the increase in BOLD activation that occurs during the first phase is no longer related to the maintenance of performance. We propose a model where local neural networks in visual cortex may be reorganized to acquire and consolidate learning, and, once this processes is completed, performance levels may be maintained without the need for further reorganization and consolidation.

RESULTS

The TDT that we used, a standard task in studies of visual perceptual learning, is known to have location specificity (Karni and Sagi, 1991). As illustrated in Figure 1, experiment 1 involved six behavioral training sessions and four fMRI sessions. In each training session, we presented a textured display along with a target array that was consistently displayed in the same visual field quadrant (the upper-left visual field). Subjects (n = 6) were presented with two types of stimuli. One was a letter, either the letter "T" or the letter "L," presented at the central fixation point. The second was a target array presented in a peripheral position for a short duration. These stimuli were intended to fixate the subjects' eyes at the center of the display and to assess learning, respectively. Subjects were asked to first identify the letter and then to report the orientation of the target array (horizontal or vertical). In the interval after presentation of the target array, a mask was presented; we refer to this interval as the stimulus-to-mask-onset asynchrony, or SOA. We employed various SOAs in the training sessions to obtain a psychometric function for determining an 80% threshold SOA. Shortening of this threshold after

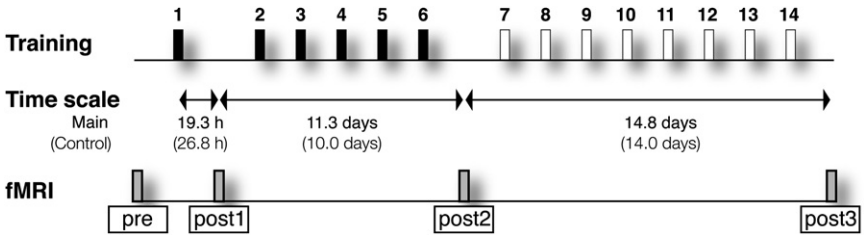

Figure 1. Experimental Design

We conducted four fMRI sessions for experiments 1 and 2. Experiment 1 (n = 6) involved six training sessions (each represented as a black bar), until the post2 scan session. The average time intervals (±standard errors) between the initial training session and post1, between post1 and post2, and between post2 and post3 were 19.3 (±2.7) hr, 11.3 (±0.6) days, and 14.8 (±1.1) days, respectively. In experiment 2 (n = 5), subjects completed eight additional training sessions (each represented as a white bar) between the scanning sessions at post2 and post3. The average time intervals between the initial training session and post1, between post1 and post2, and between post2 and post3 were 26.8 (±1.3) hr, 10.0 (±0.4) days, and 14.0 (±0.4) days, respectively, in experiment 2. Experiment 3 (n = 4) consisted of the same numbers of training and "fMRI sessions" as experiment 1. Unlike experiment 1, however, the "fMRI sessions" in experiment 3 were conducted in a mock MR scanner, with no BOLD signal measurement.

repetitive performance was considered an indication that PL had taken place (Figure 2A).

In addition to the training session, subjects took part in four separate imaging sessions, during which we acquired fMRI measurements of brain activation with a 3 Tesla MR scanner and assessed task performance during scanning (for details see Experimental Procedures). Scans were acquired before the start of training (pre-training), 10–25 hr (next day) after initial training (post-training 1; post1), 10–14 days after initial training (post-training 2; post2), and 4 weeks after initial training (post-training 3; post3) (see Figure 1). Each fMRI session involved stimulus presentation in two locations. To estimate a location-specific training effect in V1, in the fMRI sessions we presented the target arrays not only in the trained location, i.e., where subjects were presented this stimulus during the training sessions (in the upper-left visual field), but also in an untrained location (lower-right visual field). While the two location conditions were presented in random order from trial to trial, in an event-related fMRI paradigm, SOA was held constant at 100 ms, as determined from our preliminary data.

Figure 2A shows the threshold SOAs observed in the behavioral training sessions. The threshold SOA reached asymptotes in 5–6 days, corresponding to the original literature (Karni and Sagi, 1991). Figure 2B indicates that, while performance improvement was observed for the trained location [per ANOVA with repeated-measurement, $F(3,15) = 17.28$, $p < 0.001$; post hoc t tests for post2 versus pre-training, $p < 0.003$; post3 versus pre-training, $p < 0.03$], no significant improvement was observed for the untrained location. Figure 2C shows location-specific performance or fMRI activation defined as $f(1, j)/f(1, 0) - f(0, j)/f(0, 0)$, where $f(i, j)$ represents fMRI performance or BOLD signal in location i (0 = untrained location, 1 = trained location) and in post j (post0 = pre-training session, post1, 2, 3 = post-training session), respectively (see Figure S1 available online for BOLD signal changes).

Phase effects in both performance and fMRI activation were significant (ANOVA, $p < 0.01$, $p < 0.05$, respectively) in experiment 1. Location-specific performance measured in both post2 and post3 was significantly higher than in the pre-training stage (post hoc t tests, $p < 0.01$, $p < 0.05$, respectively).

To our surprise, however, location-specific fMRI activation in V1 (Figure 2C), which was boosted at post1 and post2 (post hoc t test; $p < 0.01$ and $p < 0.05$ for pre- versus post1 and pre- versus post2, respectively), decreased to the baseline level (defined as location-specific activation in V1 in the pre-training session) at post3 ($p < 0.70$ for pre- and post3). We identified a significant quadratic trend [$F(1,5) = 8.9$, $p < 0.05$], which provided evidence of two distinct patterns of dynamic relationships between performance enhancement and neural activation changes in V1, present at different stages during the time course of PL. For the initial few weeks after the onset of training, performance improved and activation increased in V1. After that period, V1 activation enhancement vanished, while the improved performance was maintained.

Note that a consistent SOA (100 ms) was used throughout fMRI sessions to evaluate and compare brain activation and performance. A relatively short SOA, which increases the difficulty of the task, had to be used to avoid a ceiling effect at later stages, such as the post2 and post3 scanning sessions. Although the correct response ratio was low in the trained location in the post1 fMRI session (Figure 2B), it does not necessarily indicate that learning did not occur from the pre-training to post1 sessions. Figure 2A shows that the 80% threshold SOA on training day 2 was significantly lower than that on training day 1, indicating that significant learning indeed occurred between training day 1 and day 2. There was a high correlation ($r = 0.82$) between the degree of SOA threshold improvement from day 1 to day 2 and the degree of change in location-specific fMRI activation from the pre-training to post1 scanning sessions (see Figure S2 for correlations between threshold SOA changes and MRI signal changes).

Reduction in V1 activation in experiment 1 was noted after training was terminated, that is, after post2. To test whether the termination of training led to this reduction of V1 activation in post3, we conducted a control experiment, experiment 2, in which a new group of subjects (n = 5) underwent continued training between post2 and post3 (Figure 1); all other conditions were identical to those used in experiment 1. We hypothesized that, if termination of training indeed led to reduced V1 activation at post3, we would not see reduced V1 activation at post3 in experiment 2. However, Figure 2D shows a trend that is generally the same as that observed in experiment 1, which allowed us to conclude that V1 activation reduction is not attributable to the termination of training (see Figure S3 for other behavioral data in experiment 2, and Supplemental Data Text 1 for the discussion of V1 and performance indices in experiments 1 and 2).

We analyzed the reaction times for the trained and untrained locations in the fMRI experiments. Figure 3A shows reaction

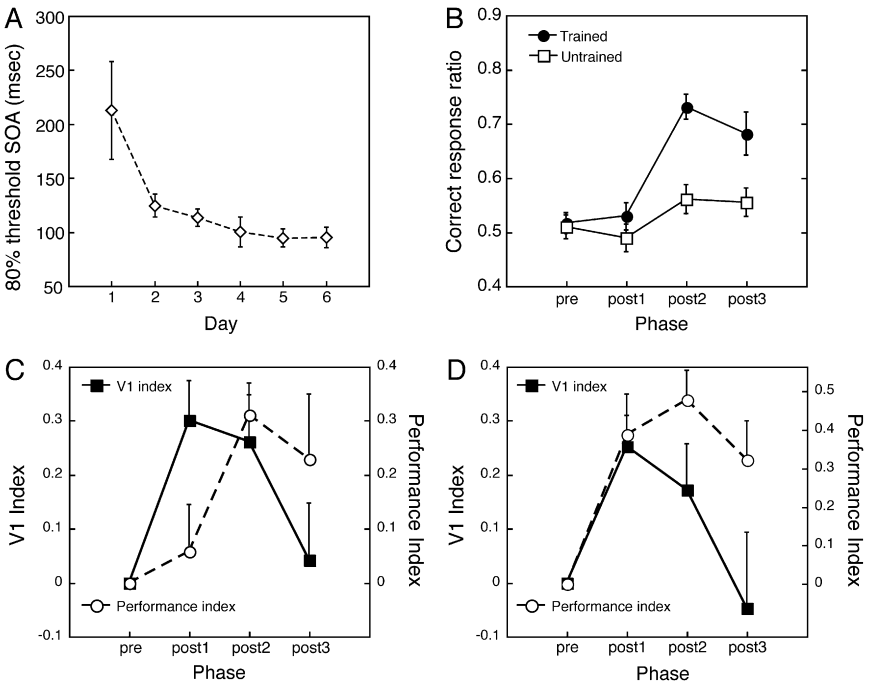

Figure 2. Results

(A) The averaged threshold SOA (±standard errors) across all subjects in experiment 1.

(B) Mean performance (±standard errors) in the trained (filled circles) and untrained (open squares) locations in experiment 1.

(C and D) Mean location-specific learning indices (+ a standard error) for the fMRI response in V1 as "V1 index" (filled squares) and for performance as "performance index" (open circles with dashed lines) in experiment 1 (C) and experiment 2 (D).

times recorded in response to the texture orientation task in experiment 1. The results of two-way ANOVA (for phase and location) with repeated measurement showed a significant phase effect ($p < 0.01$) but did not show a significant location effect. Post hoc t tests showed that there were significant differences between the pre-training and post2 scan sessions for both the trained location ($p < 0.01$) and untrained location ($p < 0.01$) and between pre-training and post3 for both the trained location ($p < 0.001$) and untrained location ($p < 0.002$). We saw the same trend for the reaction times in experiment 2 (Figure S4A).

Did the activated region size in V1 in experiment 1 change over time? Figure 3B shows the activated region size in V1. The results of two-way ANOVA with repeated measurement (for phase and location) showed no significant difference for either factor (see Figure S4B for the activated size in V1 in experiment 2).

We analyzed subject performance on the fixation letter task over time. Figure 3C shows the correct response ratio for the fixation letter task in experiment 1. The results of ANOVA with repeated measurement showed no significant effects for phase. The ratios were consistently high throughout training, indicating that subjects fixated very well during experiments. The correct response ratio for the fixation letter task in experiment 2 showed the same trend (Figure S4C).

In a further experiment, experiment 3, we measured four subjects' reaction times (RTs) and accuracy on the letter task; "fMRI sessions" were conducted in a mock scanner so that only task performance was evaluated. All other conditions were the same as in experiment 1. The results of one-way ANOVA with repeated measurement applied separately to the RTs and accuracy data of experiment 3 did not show significant effects for either RTs or accuracy (Figure S5). These results suggest that performance on the central fixation task remained constant and that the performance benefit for the peripheral task was not due to a differential allocation of attentional resources across the different fMRI sessions.

Do other brain areas—including other visual areas, such as V2 and V3, and higher cognitive areas, such as the intraparietal sulcus, superior parietal gyrus, and middle frontal gyrus—show activation changes during the time course of learning similar to those in V1? Since ANOVA indicated no significant difference between experiments 1 and 2, we combined these data and applied ANOVA with repeated measurement (phase) to indices for each of these other brain areas. Except for V1, no significant differences were found in any of these areas (Figure 4). Furthermore, we conducted various other statistical tests to examine V1 selectivity in the development of PL (see Supplemental Data Text 2 for details). Based on the tests, we conclude that no clear results were obtained to support a trend in any area other than V1.

Can the fMRI activation drop in V1 from post2 to post3 be attributed to modified attention? We observed no significant differences in either correct response ratios (Figure 2B) or RTs for orientation tasks (Figure 3A) between post2 and post3. Therefore, there was no evidence for a change in the task load. Furthermore, as mentioned previously, there was no significant change in fMRI activation throughout the time course of learning in any area other than V1. Thus, there is no clear evidence that the drop in V1 activation from post2 to post3 was due to modified attention.

DISCUSSION

The present study shows different patterns of BOLD signal and performance changes in a long time course of PL. In the initial stage, both BOLD signal and performance increase. However, in the second stage that occurs after performance saturation, the BOLD signal decreases to the level seen before training.

We propose a two-stage model for the development of PL, based on the present results and the close coupling of BOLD signal to synaptic activity (Logothetis et al., 2001; Viswanathan and Freeman, 2007). In our model, the initial training stage produces an increase in the number or strength of synaptic connections; these synapses both enhance performance and increase the fMRI signal. Note that with 100 ms SOA, we observed neither

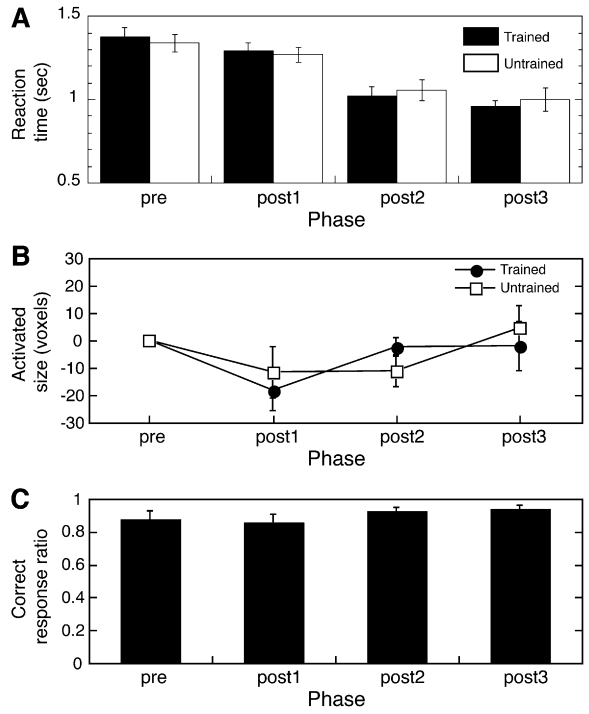

Figure 3. Reaction Time to the Orientation Task, Activated Region Size, and Correct Response Ratio for the Fixation Letter Task in Experiment 1

(A) The reaction time measured for the orientation task was defined as the time interval from the onset of the target stimulus to the button press to report array orientation. Black and white bars represent the averaged reaction time (±standard error) for the trained and untrained locations, respectively.

(B) Activated region size is defined as the mean number of voxels activated ($p < 0.01$) within the V1 cortical quadrants that corresponded to the trained (or untrained) visual field quadrants while subjects performed the TDT at the trained (or untrained) location in the pre-training session, subtracted from that in the post1, post2, and post3 scan sessions. Black circles and white squares represent the activated region size in the trained and untrained V1 cortical quadrants, respectively. Vertical bars represent standard errors.

(C) Mean correct response ratio (±standard error) for the fixation letter task.

improved performance for the trained location (Figure 2B) nor location-specific performance improvement at post1 (Figure 2C); however, trained location-specific fMRI activation was enhanced at post1, and the largest degree of performance improvement was observed on training day 2 (Figure 2A). One possible explanation is that synaptic increase/strengthening occurs gradually during the initial weeks of training and, while this increase/strengthening was sufficient to decrease the SOA threshold by training day 2 and to increase trained specific fMRI activation at post1, it was not sufficient for performance to increase before post2 with the short, fixed 100 ms.

What underlying mechanism is suggested for the second stage? The findings of this study might also be explained by synaptic downscaling. After performance becomes saturated, the number and/or strength of overall synapses may be reduced or downscaled (Censor et al., 2006; Tononi and Cirelli, 2003). However, only those synapses that are most critical to the task survive such downscaling. If the degree of BOLD activation is indicative of the degree of synaptic activity (Logothetis et al., 2001; Viswanathan and Freeman, 2007), the reduced BOLD activation that we observed is in accord with synaptic downscaling.

Our model may reconcile a controversy regarding V1 activation associated with PL. While a number of studies have found increases in V1 activation (Furmanski et al., 2004; Schwartz et al., 2002; Walker et al., 2005), some other studies have not (Ghose et al., 2002; Schiltz et al., 1999; Schoups et al., 2001). By measuring brain activity during a long time course of PL, we found that there are two distinct patterns of dynamic relationships between performance enhancement and neural activity changes in V1 at different stages of the PL time course. It may be possible that this controversy seen in the literature actually reflects findings acquired at different stages in the development of PL.

It should be noted that the activated region size did not expand in the trained V1 as learning proceeded in our study. The absence of such expansion in the activated region suggests that learning and reorganization were localized. Interestingly, these results are in contrast to the results of a study of motor skill learning (Karni et al., 1995), which indicated that an initial rapid reduction in the size of the fMRI activated region was followed by expansion. Such contrasting results may be related to differences between the modalities and tasks employed in the two studies. In the present study, we used a visual task to test plasticity in visual cortical areas, including V1, which has a highly retinotopic structure. Learning of the texture discrimination task used here is highly specific to location, which suggests that the learning involves a highly localized network (Karni and Sagi, 1991). Thus, learning and synaptic changes may occur within such a localized network.

There is a possibility that not all aspects of behavioral improvement between the post1 and post2 fMRI sessions are due to learning. Our study involved the TDT with relatively long sessions of 1520 trials. Recent results acquired with this task show that the amount of practice during a session can strongly affect performance, in the way that a larger number of trials can cause fewer improvements (Censor et al., 2006; Ofen et al., 2007). Such an effect may be due to suppressive processes related to adaptation in the visual system (Censor et al., 2006; Ofen et al., 2007). Thus, one might suggest that because the SOA in our study was gradually changed from high to low within a training session, a lower staring point may produce lower thresholds and too much adaptation can decrease performance in the next training session. The starting SOAs in the training sessions conducted between the post1 and post2 fMRI sessions (days 2–6) were indeed shortened, and, therefore, the above possibility cannot be entirely ruled out. At the same time, 1 or 2 day intervals separated one training session from the next, and it has been reported that suppressive processes may be largely eliminated during sleep that follows training (Censor et al., 2006). Variable suppression may account for the behavioral results, but not for the fMRI results in our study, as we used a constant number of trials and a constant SOA in all fMRI sessions. Thus, performance improvement between the post1 and post2 fMRI sessions may be largely attributed to learning, if not completely so.

In summary, in the present study, we measured BOLD activation and performance during a long time course of PL and have found that the shapes of the dynamics of BOLD activation and

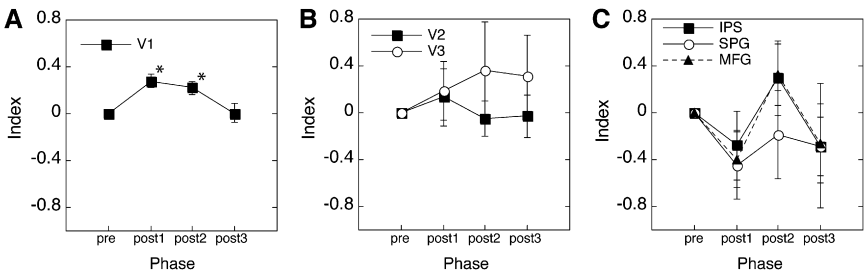

Figure 4. Trained Location Indices of Various Brain Regions

The mean activation indices (±standard error) for fMRI responses in V1 (A), V2 and V3 (B), IPS (the intraparietal sulcus), SPG (the superior parietal gyrus), and MFG (the middle frontal gyrus) (C), from the combined results of experiments 1 and 2. IPS and SPG are parts of the parietal lobe, and MFG is a part of the prefrontal area. An asterisk indicates that the index is significantly larger than the baseline ($p < 0.05$).

performance differ. During the initial few weeks of training, trained location-specific activation in V1 increased, as did task performance. However, after performance increase became saturated, performance enhancement was maintained, while activation increase disappeared. These results are in accord with our proposed model of plasticity, in which different patterns of synaptic activity occur at different stages. Future studies will be required to address whether the proposed two-stage model could be generalized to other types of PL in the visual system and to other sensory modalities.

EXPERIMENTAL PROCEDURES

Subjects

A total of 15 subjects (8 females and 7 males) with normal or corrected-to-normal vision were employed. Six subjects (age range: 29–36 yr, 3 females and 3 males) participated in both the behavioral training and fMRI sessions of experiment 1. Five subjects (22–39 yr, 2 females) in experiment 2. Four subjects (22–28 yr, 3 females) in experiment 3. All subjects gave written informed consent for their participation in the experimental protocol approved by the Institutional Review Boards of the Massachusetts General Hospital and Boston University.

Behavioral Training Session

With their chin and forehead fixed, each subject viewed visual displays on a screen positioned 57 cm from their eyes. All behavioral experiments were conducted in a dimly lit room. We employed a texture discrimination task that has been widely used in visual perceptual learning studies. In each TDT trial, we briefly presented a test stimulus (13 ms) that was followed by a blank screen (presentation period varied by trial) and a mask stimulus composed of randomly oriented V-shaped patterns (100 ms); the mask stimulus was presented in the interval after target array presentation (stimulus-to-mask-onset asynchrony, SOA). The test stimulus consisted of a centrally located letter, either "T" or "L," and a peripherally positioned horizontal or vertical array of three diagonal bars (target arrays) on a background of horizontal bars. While keeping their eyes fixated on the center of the visual field, subjects were asked to respond twice for each trial: once to identify the letter and once to indicate the orientation (horizontal or vertical) of the target array. The fixation letter task was intended to ensure the subjects' focus and fixation at the center of the visual field; the target array discrimination task was used as a measure of perceptual learning. Each line segment of the peripheral target array was arranged within a 19 × 19 lattice in the area of a 14° × 14° visual angle. Lines were 0.43° × 0.07° and spaced 0.7° apart. The position of each line segment was jittered slightly, by 0°–0.05°, from trial to trial. The position of the target array also varied randomly from trial to trial, but was consistently presented within a specific quadrant (see below) and within a 2.95°–5.15° visual angle from the center of the display. All line segments were gray (32 cd/m^2) and presented on a black (0.5 cd/m^2) background. Immediate auditory feedback was given only for the fixation letter task to facilitate subjects' fixation. No feedback was given for the orientation task in this experiment following the order to adhere to the original task procedure developed by Karni and Sagi (Karni and Sagi, 1991) and because learning occurs without feedback in the TDT (Karni and Sagi, 1991; Sagi and Tanne, 1994).

During the training sessions, the horizontal or vertical target array was presented only in the upper-left visual field quadrant. Each training session contained 1520 trials, presented in 40 blocks. Each block contained 38 trials with a constant SOA. Each training session started with a block with a longer SOA, for instance 250 ms, which was decremented by 20–40 ms every two to four blocks. As training proceeded, the SOA in each block was shortened, thus increasing the difficulty of the task. An initial SOA was determined daily and individually based on earlier performances. The percentage of correct responses was calculated for each SOA in order to construct a psychometric function for determining the threshold SOA, at which subjects reach 80% correct responses by interpolation.

Subjects took part in training sessions once every 2 or 3 days. Experiments 1 and 3 involved six training sessions. Experiment 2 included an additional eight sessions, thus a total of 14 sessions.

fMRI Experiments

Scanning sessions were conducted on four separate occasions: at the pretraining, post1, post2, and post3 sessions shown in Figure 1. Stimuli were generated on a Mac G4 and presented via LCD projector (Sharp Note Vision 6). The fMRI experiments involved two location conditions for presentation of the target arrays, which were displayed in random order in either the upper-left visual field (trained condition) or lower-right visual field (untrained condition), using an event-related fMRI paradigm. In the event-related paradigm, the timing for the presentation of each condition was calculated with optseq2 software (Dale, 1999; Dale et al., 1999b) to randomize the interstimulus interval from trial to trial for maximized the statistical efficiency.

We presented 128 trials for each of the two locations during each fMRI session; a single trial lasted 2 s. At the beginning of each trial, a blue or green fixation cross was presented for 500 ms, followed by a blank screen for 250 ms. The color of the fixation cross served as a cue for the location of a target array to follow. A blue fixation cross indicated that the target array would appear in the upper-left (trained) quadrant; a green cross indicated that the array would appear in the lower-right quadrant. A target texture was then presented for ~20 ms (the temporal resolution limit of the display), followed by a mask for 100 ms; the SOA was a constant 100 ms. As in the behavioral training session, subjects were asked to respond to the fixation and orientation tasks by pressing a button on a box that they held in their hand. Immediate auditory feedback was given only for the fixation letter task, to facilitate subjects' fixation.

Image Acquisition

Subjects were scanned in a 3T MR scanner (Allegra or Trio, Siemens); a head coil was used throughout the experiments. Functional MR images were acquired using gradient echo EPI sequences (TR = 2 s, TE = 30 ms, flip angle = 90°) for measurement of BOLD contrast. Thirty-five contiguous slices (3 × 3 × 3.5 mm^3) oriented parallel to the AC-PC plane were acquired to cover the entire brain.

All functional data were registered to the individual anatomically reconstructed brain (Dale et al., 1999a; Fischl et al., 1999). For the anatomical reconstruction, we acquired three T1-weighted MR images (MPRAGE) (TR = 2.531 s, TE = 3.28 ms, flip angle = 7°, TI = 1100 ms, 256 slices, voxel size = 1.3 × 1.3 × 1.0 mm^3, resliced during analysis to 1 mm^3). This same anatomical reconstruction was used for brain parcellation to localize individual gyri and sulci (Fischl et al., 2004).

Definition of Region of Interest

Four retinotopic quadrants (upper left, upper right, lower right, and lower left of the visual field) of V1, V2, and V3/VP areas were localized individually in a separate fMRI session that used a standard flickering checkerboard pattern (Engel et al., 1994; Fize et al., 2003). In addition, eccentricity was localized individually by using annulus stimuli of various sizes. In the subsequent analysis, we used regions of 3°–5° of eccentricity in V1, V2, and V3/VP. Because the retinotopy of V4 is controversial (Wandell et al., 2005), V4 was not included in our analysis.

The middle frontal gyrus, superior parietal gyrus, and interparietal sulcus were identified individually using the brain parcellation method (Fischl et al., 2004).

fMRI Data Analysis

Data were analyzed with FS-FAST and FreeSurfer (http://surfer.nmr.mgh.harvard.edu) software. All functional images were motion corrected (Cox and Jesmanowicz, 1999), spatially smoothed with a Gaussian kernel of 5.0 mm (FWHM), and normalized individually across scans. In this normalization process, the mean intensity for the entire functional volume was computed for each scan. The global mean of the entire brain was rescaled so that the same mean was set across scans. A finite impulse response model (Burock and Dale, 2000) was employed to estimate hemodynamic response (time course) to each condition (trained or untrained locations) in each ROI, in 20 1 s interval time points. The time courses for each condition, for each ROI, were then converted into percent signal changes, by subtraction and division of the mean value of the ROI. Note that the mean value of each ROI, which is a part of the entire functional volume, was not assured to be the same across the different fMRI sessions.

To compute a location-specific response index in V1, we first normalized the peak hemodynamic response (4–6 s from stimulus onset) at each session by dividing it by the peak response in the pre-training session. The normalized value of the upper-left V1, which corresponded to the untrained location, for the untrained condition, was then subtracted from the normalized value of the lower-right V1, which corresponded to the trained location, for the trained condition at each session, respectively. Response indices for V2 and V3 were computed in the same way as for V1. Response indices for the middle frontal gyrus, superior parietal gyrus, and intraparietal sulcus were computed by subtracting the normalized value of the left hemisphere of each region in the untrained condition from the normalized value of the right hemisphere of each region during the trained condition.

SUPPLEMENTAL DATA

The Supplemental Data for this article can be found online at http://www.neuron.org/cgi/content/full/57/6/827/DC1/.

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